

Case Report

Myocardial infarction in association with misuse of anabolic steroids

Christine Kennedy

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Abuse of androgenic and anabolic steroids is becoming both a medical and public health problem. It is generally underestimated just how prevalent steroid abuse is amongst young sports people. Vascular thrombosis (myocardial infarction,¹ stroke,² arterial thrombosis in a limb³) has been documented in the USA in steroid abusing athletes in the past decade. The problem is now appearing in Britain, and two cases of myocardial infarction and one of pulmonary embolus associated with steroid abuse have been documented since 1990.

CASE REPORT. A 24 year old competitive bodybuilder presented three days after development of recurrent, dull central chest pain radiating to his left arm. He had been aware of similar, less severe episodes over the previous six weeks, which he had attributed to muscular strain after heavy weight-training. He spontaneously admitted to use of anabolic steroids for two years — taking multiple steroid preparations in mixed dosage forms, both orally and by intramuscular injection, at high doses for a prolonged period — a process called “stacking and cycling” by those involved.⁴

Over the last six week cycle he had been taking oral stanozolol 40 mgs daily, nandrolone 200 mgs intramuscularly twice weekly, and Sustanon 250 (testosterone esters) 1 ml intramuscularly once a week. He also smoked 30 cigarettes daily — a 10 year habit. There was no family history of heart disease or lipid disorder.

Serial electrocardiographs and cardiac enzyme measurements confirmed lateral myocardial infarction. Serum creatine kinase (myocardial subfraction) rose from 42 to 68 units in 24 hours and fell to 33 by 48 hours. The percentage of this enzyme rise increased from 6 to 14%. (More than a 7% rise is diagnostic of myocardial necrosis). Fasting lipid measurements showed a raised serum total cholesterol (8.5 mmol/l) and low HDL cholesterol (0.7 mmol/l), giving a ratio of 12:1 (the normal ratio is less than 6:1). Fasting serum triglyceride was 1.65 mmol/l (normal range 1.2 — 3.0 mmol/l).

Echocardiogram showed a normal sized heart with good left ventricular function and a small area of apical infarction. Exercise stress testing under the modified Bruce protocol gave a total exercise time of 17 minutes, without chest pain and

The Waveney Hospital, Ballymena, Co. Antrim.

Christine Kennedy, MB BCH BAO, Senior House Officer.

the test was terminated due to fatigue. Blood pressure and pulse rate rose with exercise as would occur in a normal healthy individual. At submaximal exercise he developed frequent ventricular ectopic beats with couplets and bigeminy. These ectopic beats decreased at peak exercise and recurred again in the early recovery period. He also developed ST segment elevation in standard lead 1 and the anterolateral chest leads, which reverted to normal after six minutes.

His blood pressure remained intermittently elevated (maximum 195/110 mmHg) and he was treated with atenolol 100 mg daily and aspirin. He was not started on a cholesterol-lowering agent as the lipid profile should improve once the anabolic steroids are discontinued. He was advised to stop all steroid abuse, to stop smoking, to avoid weight-training, and was encouraged to do moderate aerobic exercise. He is awaiting coronary angiography.

DISCUSSION

Although hypertension and adverse lipid profiles are well established side effects of anabolic steroid overdose, acute thrombosis has been linked only recently to androgen abuse. There is no direct evidence that these steroids are thrombogenic but experimental data suggests they probably facilitate thrombosis by an effect on platelet aggregation and on coagulation proteins.⁵⁶⁷ In this case the aetiological factors favouring thrombosis would seem to be the adverse lipid profile and hypertension conferred by use of steroids; anabolic exercise (weight training in itself imparts a negative effect on lipids which is amplified when abusing steroids); cigarette smoking; and the possibility of steroids affecting platelet function and the clotting factors. At a molecular level steroid abuse produces mitochondrial and myofibrillar changes similar to those seen in early heart failure.⁸ In athletes who die suddenly there is a higher incidence of right ventricular cardiomyopathy, although there is no direct evidence to link this to steroid abuse. Echocardiographic studies show concentric left ventricular hypertrophy and altered pressure-volume relationships within the heart, but in comparative studies between steroid-users and non steroid-users no statistically significant difference in left ventricular function could be found.⁹

It is well known that serious side-effects can be attributed to anabolic steroids. These may occur within weeks (decreased reproductive function, altered serum transaminases or deleterious lipid profile) or take years to develop, such as hepatocellular carcinoma. All the early side-effects are reversible provided the drug abuse is discontinued. Greater public awareness of the problem of steroid misuse is necessary. By and large, the medical community do not recognise the wide availability of the compounds or the prevalence of their misuse.

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